


Childhood Socioeconomic Status and Stress in Late Adulthood: A Longitudinal Approach to Measuring Allostatic Load

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Abstract

Objectives: This study examines how the effects of childhood socioeconomic status (SES) may carry on into late adulthood. **Methods:** We examine how childhood SES affects both perceived stress and allostatic load, which is a cumulative measure of the body's biologic response to chronic stress. We use the National Social Life, Health, and Aging Project, Waves 1 and 2, and suggest a novel method of incorporating a longitudinal allostatic load measure. **Results:** Individuals who grew up in low SES households have higher allostatic load scores in late adulthood, and this association is mediated mostly by educational attainment. **Discussion:** The longitudinal allostatic load measure shows similar results to the singular measures and allows us to include 2 time points into one outcome measure. Incorporating 2 separate time points into one measure is important because allostatic load is a measure of cumulative physiological dysregulation, and longitudinal data provide a more comprehensive measure.

Keywords

allostatic load, biomarkers, early life conditions, socioeconomic status, stress

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Introduction

Growing evidence suggests that early life experiences are relevant to health and well-being in later life. Rather than only focusing on experiences and conditions in adulthood, research should also pay attention to early life in order to fully understand health and mortality. The relationship of adult socioeconomic status (SES) with health and mortality has been well established, but evidence continues to suggest that this relationship may begin much earlier with parental SES in childhood. More specifically, socioeconomic conditions in early life have been recognized as an important factor for health and mortality risk in later life.^{1–3} Children have no control over the socioeconomic class they are born into, but social forces can have a significant and lasting impact as children grow into adulthood. Children born into lower socioeconomic positions are faced with conditions (ie, lower financial resources, poorer housing) that have been shown to affect health at all ages, and decrease the possibility of upward mobility to a higher social class in adulthood.^{4,5}

The stress process model suggests that social positions, such as SES, affect many of the stressors and stressful life events an individual experiences throughout the life course.^{6,7} Repeated and chronic stressors (ie, feeling unsafe in one's own neighborhood) can affect health in a different way than intermittent stressful events (ie, feeling unsafe during a thunderstorm). Studies show that experiencing poverty consistently over time has a worse effect on health than experiences of poverty interspersed

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with times of less financial hardship.^{7,8} Recent work has focused on examining the biological processes (ie, inflammation) underlying the pathway between SES, stress, and health. More specifically, research has focused on how the chronic stress associated with low SES disrupts the normal functioning of biological systems, such as the endocrine or nervous systems, which regulate how the body functions. Physiological dysregulation results when the normal processes that regulate the body become impaired, and it increases the risk of negative health outcomes such as diabetes or high blood pressure. Allostatic load is a concept used to quantify this disruption of normal biological functioning due to chronic stress.⁹ This study aims to provide a better understanding of the role that early life conditions play in SES, perceived stress, and allostatic load in later life. Perceived stress reflects one's own feelings and ideas about how stressful their lives are, and how much control they have over that stress. The specific goals of this article are to (1) add to previous findings by examining the relationship between childhood SES and adult education, wealth, and perceived stress; (2) expand on existing literature by determining the relationship between childhood SES, adult outcomes, and allostatic load in late adulthood; and (3) provide a unique contribution to the literature by suggesting a novel method of measuring allostatic load longitudinally.

Allostatic Load

Allostasis refers to the long-term effects of stress on physiological functioning across multiple biologic systems in the body.¹⁰ Allostatic load is a compilation of biological measures, from multiple physiological systems (ie, the immune system), used to measure the extent of physiological dysregulation (from interrupted biological processes) resulting from the body's response to chronic stress.¹¹ Immediate responses to stress cause the release of primary mediators, which are meant to regulate the body's physiological response to acute stress. The primary mediators are (1) stress hormones, such as norepinephrine or cortisol, in the neuroendocrine system; and (2) anti-inflammatory cytokines from the immune system, which cause acute inflammation. If a stress response is longer term (chronic stress), the dysregulation of primary mediators (stress hormones and anti-inflammatory cytokines) causes secondary outcomes to the other biological systems.^{9,12} Secondary outcomes can affect the immune, metabolic, cardiovascular, and anthropometric systems. Examples of secondary outcomes include elevated C-reactive protein (CRP), which is a response to chronic inflammation, and increased body mass index (BMI). Tertiary outcomes are the health outcomes manifested by these changes in functioning, such as poor self-rated health and disability.⁹

Due to the cumulative nature of allostatic load, it is suggested that the relationship between SES and allostatic load be studied from a life course approach.¹³ There is evidence that allostatic load mediates the relationship between SES and mortality, and SES is inversely associated with allostatic load for children, adolescents, and adults.¹³⁻¹⁷ Both education and income show evidence of allostatic load gradients, though the benefits of education and higher income may not affect all racial identities similarly.^{17,18} Living in an impoverished neighborhood, poor housing quality, neighborhood disadvantage, perceived economic distress, and an adult moving out of the home all have evidence of lasting negative impacts on allostatic load.^{19,20}

Measuring Allostatic Load Longitudinally

Previous studies have suggested the importance of studying allostatic load longitudinally, though there are only a limited number of examples in the literature.^{9,21-23} Reasons for the lack of longitudinal allostatic load studies include difficulty collecting data and difficulty analyzing a composite measure over time. Respondents must be willing to participate in data collection involving blood or tissue samples, and then these must be processed in a laboratory. If biological data are available, then analyzing change in allostatic load over time becomes the issue. Some studies have used the difference between scores over time, but this may not properly shed light onto the cumulative allostatic load measure. For example, if one respondent has an allostatic load score of 7 at Wave 1 and 7 at Wave 2, the change would be 0. However, this respondent has a high allostatic load score in both waves, but the same change score as a person who has an allostatic load score of 0 for both waves.

Two previous studies have incorporated a longitudinal allostatic load measure by showing that increase (decrease) in allostatic load is associated with negative (positive) health outcomes. Karlamangla et al²⁴ found that increases in allostatic load were associated with increased mortality risk. A study of older adults in Taiwan observed allostatic load change over 6 years by placing respondents into groups based on changes (or lack of) in allostatic load score.²⁵ They found that both high allostatic load and the rapid increase in allostatic load score over 6 years (compared with a decline) were associated with increased mortality risk. In a similar manner, another study showed that allostatic load trajectories with more than 2 time points have been shown to predict later mortality risk, frailty, and certain chronic diseases.²⁶

Another approach used in the literature measures the rate of allostatic load change over 3 or more points in time, called growth curve models. A study of middle-aged women found that those reporting higher levels of

perceived stress have faster rates of increase in allostatic load over time.²⁷ They also found that most of their measures, such as being black and low SES, were predictive of allostatic load at one point in time, rather than rate of change. Merkin et al²⁸ also used growth curve models, separating analyses by those with low allostatic load at baseline and those with high allostatic load at baseline. For the low baseline group, those who achieved high levels of education in adulthood showed a slower rate of increase over time. They also examined the educational attainment of the respondents' parents, though this was not found to be an independent predictor of allostatic load change. The findings mentioned above show that increases in allostatic load over time can lead to greater mortality risk, more frailty, and particular health conditions.²⁴⁻²⁶ However, to our knowledge, these are the only existing studies observing allostatic load change over time. Given the lack of attention to observing allostatic load longitudinally and the implications of the few existing studies, it is important to continue exploring methods of measuring change.

Theoretical Framework

The cumulative advantage/disadvantage theory posits that disadvantage results in differential outcomes that increase over time, and this framework is used to explain socioeconomic inequalities in health.²⁹⁻³¹ The cumulative inequality theory expands on cumulative disadvantage theory and incorporates the stress process model and the life course perspective to focus on systemic inequalities and how these inequalities interact with personal trajectories to influence aging.³² These structural inequalities exist before a person is even born, with social forces shaping the environment a child is brought into.^{32,33} These inequalities accumulate over time and are thus best studied from a life course approach. This allows researchers to observe health trajectories beginning with inequalities in early life that may become even more pronounced as individuals age. For example, being born into a certain social class can provide various advantages and disadvantages for opportunities in education and occupation in early adulthood, which, in turn, affect health and well-being for the remainder of life.³⁴ Cumulative inequality emphasizes not only the importance of early life position but also takes into account human agency and psychosocial resources, citing that using these resources positively (or negatively) can reshape health trajectories.³²

Methods

Data

We use data from Waves 1 and 2 of the National Social Life, Health, and Aging Project (NSHAP) for this study.³⁵

This biosocial data set provides information on physical, mental, and social health among participants aged 57 to 85 in the United States. Wave 1 data were collected between July 2005 and March 2006, and Wave 2 data were collected between August 2010 and May 2011. There are 3005 individual cases in Wave 1 and 3377 individual cases in Wave 2. The Wave 2 interviews include 2261 Wave 1 respondent interviews. The attrition between Waves 1 and 2 is due to 430 deaths, 139 in poor health unable to participate, 4 in a nursing facility, 161 unknown but presumed to be alive, and 10 unknown. In each wave of data collection, only a subsample of respondents had all biomeasures collected, which further reduced our final sample size.

We established 2 study samples: Sample 1 was used to establish the high-risk cutoff points for the allostatic load scale, and Sample 2 was the study sample used for analyses. We felt that using a baseline sample that included respondents who died before Wave 2 follow-up would produce an allostatic load scale that accounts for some of the survival effects seen in older adults. Karlamangla et al²⁴ used a similar approach in their longitudinal study of allostatic load. Sample 1 was created using only Wave 1 data. Respondents missing race/ethnicity information (N = 13) were dropped from the sample. Cases with missing values on more than 2 of the 8 biomarkers used to measure Wave 1 allostatic load (N = 424) were deleted. Remaining missing values were imputed using the sample mean and additional respondent information for glycosylated hemoglobin (a measure of blood sugar) and CRP (a measure of inflammation). Cases with missing information on remaining key variables were dropped using listwise deletion (N = 472) for a final sample size of 2102.

Sample 2 includes only individuals interviewed in both Waves 1 and 2 (N = 2261). This is because Wave 1 did not have the information regarding childhood SES, and Wave 2 added respondent partner interviews. Cases with missing values on more than 2 of the 8 biomarkers used to measure allostatic load in Waves 1 and 2 (N = 366) were deleted. Remaining missing values were imputed using the sample mean and additional respondent information for glycosylated hemoglobin and CRP. Cases missing race/ethnicity (N = 7), childhood SES (N = 284), Wave 1 or Wave 2 perceived stress (N = 193), and total assets (N = 46) were dropped from the sample for a final sample size of 1365. Our analyses initially intended to include a measure of Wave 2 perceived stress in addition to Wave 1, but we chose instead to focus on allostatic load. We examined the 116 respondents missing Wave 2 perceived stress to add back into our sample and found that their values on key variables were significant outliers compared with our original sample of 1365 and therefore felt it best to leave those cases out of our sample.

Measures

Allostatic Load. Allostatic load is the focal outcome of this study. The allostatic load index was created using 8 biomarkers, based on availability of data and previous research. Allostatic load is intended to be a multisystem measure of the response to chronic stress; therefore, utilizing biomeasures from various physiological systems is ideal. Additionally, it is recommended that both primary mediators (which capture acute effects of stress) and secondary outcomes (which refer to more long-term impacts of stress) are included in the composite allostatic load measure.⁹ The 8 biomarkers used for our allostatic load index cover 5 different physiological systems and include 1 primary mediator and 7 secondary outcomes. Dehydroepiandrosterone (DHEA) is used in the allostatic load scale as a primary mediator from the neuroendocrine system. DHEA is theorized to act as a regulator to cortisol, a stress hormone, which indicates that low levels of DHEA are problematic.^{36,37}

The secondary outcomes used in this allostatic load index are the following: CRP from the immune system; glycosylated hemoglobin from the metabolic system; systolic blood pressure, diastolic blood pressure, and heart rate from the cardiovascular system; and waist circumference and BMI from the anthropometric system. CRP is a substance produced in the body in response to inflammation, making it a useful biomeasure of inflammation and the immune system's response to chronic stress.³⁸ Glycosylated hemoglobin is a measure of glucose in the blood, and chronically high levels are associated with diabetes.³⁹ Systolic blood pressure, diastolic blood pressure, and heart rate are all measures of cardiovascular health. High levels of these biomarkers are associated with hypertension and poor cardiovascular functioning.⁴⁰ Waist circumference and BMI are intended to measure body fat composition, with higher levels indicating more body fat and cardiovascular risk.⁴¹ CRP and glycosylated hemoglobin were both measured using dried blood spots and DHEA was measured through saliva samples. No significant differences in demographic or health characteristics were found between those who agreed to provide blood spots and those who declined.⁴²

For each of the 8 measures, individuals were coded as to whether they exhibit high risk or not, with 1 indicating high risk. High risk was determined by the using the upper quartile of the sample (lower quartile for DHEA) as in previous studies measuring allostatic load.¹³ This is the preferred method of measuring risk for allostatic load scores as clinical cut points are not established for many of the measures, in particular when looking at a specific group such as older adults.^{9,13} The

risk indicators were then summed to create the allostatic load index, ranging from 0, meaning no high-risk indicators, to 8, meaning 8 high-risk indicators.

Using the imputed mean adopts a conservative approach in that none of these missing values were assigned a high-risk score, with the exception of those who were missing glycosylated hemoglobin and answered that they were diabetic (Wave 1 N = 68; Wave 2 N = 11). CRP scores are notably higher for Wave 2; therefore, the 60 individuals missing this measure, who had the mean imputed (separately by sex) did end up being past the high-risk threshold.

Finally, in order to account for the many individuals who may be on medications affecting their allostatic load, we used medication information reported in Wave 1 to adjust the allostatic load scales accordingly. The medications data for Wave 2 were not yet available, so we used the Wave 1 medications data for both allostatic load measures. For individuals taking blood pressure medications, their systolic blood pressure indicator was changed to 1 if it was not already in the high-risk category. Similarly, for respondents taking diabetic medications, their glycosylated hemoglobin score was changed to at-risk.

Longitudinal Allostatic Load Score Over 5 Years. This score used the scale created from measuring the change between risk scores of individual biomarkers between waves. While it may seem like simply taking the difference between allostatic load scores for each time point would be sufficient, this does not accurately reflect risk. If a person has the same score for both time points, their change score would be 0, whether or not they had a low score of 1 for each time point, or the highest risk score for both time points. We began by using the dichotomous risk indicator for each biomarker for both time points. Each biomarker is given a score between 0 and 3, with 3 indicating the most risk. If the individual had a value of 0 for the risk indicator at both time points, their score was 0. If the value of the risk indicator decreased from 1 to 0 between time points, the score was 1. If the value of the risk indicator increased from 0 to 1 between time points, the score was 2. If the value of the risk indicator was 1 for both time points, the score was 3. The scores for each of the 8 biomarkers were then summed. The change scores ranged from 0 to 21, but the highest potential score using the scale was 24, which indicates having a score of 1 for each of the 8 biomarkers at both time points. It may be considered that decreasing risk between waves indicates less risk, but not being at risk in either wave should theoretically have less cumulative biological impact. The allostatic load scores from Waves

1 and 2 used to create this measure were both adjusted for medications.

Childhood SES. Childhood SES was a focal predictor variable that was measured using responses to the question: "During the time from about age 6 to age 16, would you say your family was very well off financially, fairly well off, about average, not so well off, or not well off at all?" If respondents considered family finances growing up as "not so well off" or "not well off at all," they were coded as 1 for family financial background, otherwise they were coded as 0. There were very few respondents who reported very well off or fairly well off and the goal of this article is to observe the outcomes of socioeconomic disadvantage in childhood; therefore, we felt the dichotomous indicator is best.

SES in Adulthood. Educational attainment was assigned to 1 of 4 groups based on degrees earned: less than a high school diploma, high school graduate, some college, and bachelor's degree or higher. A set of dummy variables was created using less than a high school diploma as the reference group. For education as an outcome, dichotomous measures were used for each of the education groups for 4 separate odds ratio models.

Household assets was used as a measure of late adult SES in that many of the respondents are retired and therefore income may not be an accurate reflection of SES.¹³ Respondents were asked,

Now I'd like you to think about all of the assets of your household. These are things like your house (if you own it), cars, other rental properties and businesses you own, and financial assets like savings accounts, stocks, bonds, mutual funds, and pensions. Altogether, how much would you say that amounted to, approximately, after accounting for the loans you might have to pay off?

Answers range from \$0 to \$20 million. Those who answered "don't know" or refused an answer ($N = 414$) were then asked a series of follow-up questions to determine an approximate value range of their assets. There were 5 categories that the follow-up questions presented: less than \$10 000 total; \$10 000 to \$49 999; \$50 000 to \$99 999; \$100 000 to \$499 999; and \$500 000 and higher. We used these follow-up questions to impute assets for those who responded to the follow-up questions. If total assets was still missing, the Wave 2 value was used if present. The remaining missing cases were deleted ($N = 46$). Finally, because the distribution of assets is so skewed, the logged value is used for analyses.

Perceived Stress Scale. A perceived stress scale was created using the 4 question version of the original 14-item

questionnaire by Cohen et al.⁴³ The purpose of this scale is to measure how stressful one interprets his or her life situations to be. For this study, its purpose was to measure psychosocial functioning and response to stressors. For each of the 4 questions, respondents were asked how frequently they had experienced the following in the past week: "I felt that things are going my way," "I felt that difficulties were piling up so high that I could not overcome them," "I was unable to control important things in my life," and "I felt confident about my ability to handle personal problems." Responses were chosen from the following: "rarely or none of the time," "some of the time," "occasionally," and "most of the time." We assigned values ranging from 0 to 3 for each response, with 3 indicating the most stress experienced. We reverse coded the positive questions for a total scale of 0 to 12, with 12 indicating the highest level of perceived stress. For perceived stress as an outcome, we used a dichotomous version of the variable to indicate "high perceived stress." If a respondent's perceived stress score was 3 or greater, they were coded "1" for high stress. The distribution of perceived stress scores did not have good model fit as an ordinary least squares regression continuous outcome or a count outcome, therefore we felt the dichotomous outcome had the best fit.

Controls. Age, sex, race/ethnicity, and marital status were used as control variables. Age was controlled for as a continuous variable, ranging from 57 to 85 in Wave 1. Sex was recoded as the dummy variable "female" where "1" indicates female and "0" is male. Race/ethnicity was assigned based on participant self-report selected from 1 of 4 categories: non-Hispanic White, non-Hispanic Black, Hispanic, or other. We used this categorical variable to create a set of 4 dummy variables, using non-Hispanic White as the reference group. Marital status, also taken from Wave 1, was coded as a dichotomous variable, married coded as 1, and unmarried as the reference group. Unmarried takes into account any response that is not listed as "married."

Statistical Analyses

We used Stata 13 for all data cleaning and analyses. The first part of our analyses focused on examining how childhood SES affects adult outcomes (education, wealth, perceived stress). Logistic regression was used to determine the odds ratios for 4 levels of educational outcomes (less than high school, high school diploma, some college, and college) and to determine the relationship between childhood SES and perceived stress in later adulthood. Ordinary least squares regression was used to determine the relationship between childhood SES and total household assets in late adulthood.

Table 1. Descriptive Statistics for the Full Sample and by Childhood SES^a.

Wave 1 Measure	Range	Full Sample, Mean/%	Low CSES, Mean/%	Not Low CSES, Mean/%
N	—	1,365	634	731
Total assets	\$1.00-\$20 million	\$653 089 (\$1 577648)	\$496 621*	\$788 795
Logged assets	0-16.81	12.11 (2.17)	11.83*	12.36
Perceived stress	0-12	1.59 (2.19)	1.78*	1.43
W1 AL score	0-8	2.30 (1.63)	2.43*	2.18
W2 AL score	0-8	2.41 (1.60)	2.53*	2.31
Total AL score	0-21	7.12 (4.45)	7.49*	6.80
Age	57-85	67.7 (7.40)	68.3*	67.3
Sex				
Female	0-1	49.7%	50.5%	50.1%
Male	0-1	50.3%	49.5%	49.9%
Race				
White	0-1	77.4%	74.1%*	80.2%
Black	0-1	10.8%	14.2%*	7.9%
Hispanic	0-1	9.2%	9.5%	8.9%
Other	0-1	2.6%	2.2%	3.0%
Education				
Less than high school	0-1	14.7%	20.0%*	10.1%
High school graduate	0-1	24.4%	26.0%	23.0%
Some college	0-1	33.2%	32.3%	33.9%
College degree	0-1	27.7%	21.6%*	33.0%
Marital status				
Married	0-1	66.6%	64.5%	68.4%
Not married	0-1	33.4%	35.5%	31.6%
Childhood SES				
Not low	0-1	53.5%	—	—
Low	0-1	46.5%	—	—

Abbreviations: SES, socioeconomic status; CSES, childhood SES; W1, Wave 1; W2, Wave 2; AL, allostatic load.

^aTable data are taken from the National Social Life, Health, and Aging Project, Waves 1 and 2. N = 1365. Standard deviations are in parentheses.

*Indicates significant difference between low family income group and average family income, $P < .01$.

The next part of our analyses focused on direct and indirect effects of childhood SES on late adulthood allostatic load at 2 singular time points (Wave 1 and Wave 2). Poisson regression models were used to assess the relationship between childhood SES and allostatic load in both Waves 1 and 2. We used the “countfit” program (downloadable for Stata) that compares various fit statistics in order to determine that Poisson models were preferred for this count distribution. The final step of analyses used both allostatic load measures in order to observe allostatic load change as a focal outcome. Using the “countfit” program, we determined that negative binomial regression was a better fit for the distribution of this new outcome. For all Poisson and negative binomial regression models, incidence rate ratios are presented, and we use the “vce(robust)” option in order to control for potential minor violations of underlying assumptions.

Results

Table 1 provides descriptive statistics for the sample as a whole (N = 1365), and bivariate statistics by childhood SES. A little more than half of respondents (53.5%) report having average or above-average income in childhood, and 46.5% report lower than average income growing up. The bivariate statistics suggest more disadvantaged outcomes for the low childhood SES individuals.

Multivariate Results

Adult SES. Table 2 shows odds ratio coefficients for each separate level of educational attainment as its own dichotomous outcome. Low childhood SES individuals have significantly greater odds of being in the lowest educational achievement group, compared with those of

Table 2. Odds Ratios of Educational Attainment^a.

	Less Than High School	High School Diploma or Equivalent	Some College	College Graduate
Low childhood SES	2.18*** (0.38)	1.16 (0.15)	0.95 (0.11)	0.60*** (0.08)
Age	1.05*** (0.01)	1.03** (0.01)	0.99 (0.01)	0.96*** (0.01)
Female	0.82 (0.14)	1.24 [†] (0.16)	1.60*** (0.19)	0.52*** (0.07)
Black	4.33*** (0.95)	0.84 (0.17)	0.86 (0.16)	0.46*** (0.11)
Hispanic	13.43*** (2.97)	0.36*** (0.10)	0.66 [†] (0.14)	0.27*** (0.08)
Other	0.34 (0.35)	0.58 (0.26)	1.39 (0.48)	1.34 (0.48)

Abbreviation: SES, socioeconomic state.

^aTable data are taken from the National Social Life, Health, and Aging Project, Waves 1 and 2. Standard errors are in parentheses. N = 1365.

[†]P ≤ .10. *P ≤ .05. **P ≤ .01. ***P ≤ .001.

average or above socioeconomic backgrounds. Those with low childhood SES have over 2 times the odds of reporting less than a high school diploma as their highest level of educational attainment. For the highest level of educational attainment, college degree or higher, having low childhood SES significantly decreases the odds of attaining a college degree by 40%. Table 3 shows ordinary least squares regression coefficients for wealth (total logged assets) and odds ratios for high perceived stress. Low childhood SES is a significant predictor of decreased assets in late adulthood in Model 1, but this association is no longer significant once education is added in Model 2. Model 3, the full model, shows significant wealth advantages of the 3 education levels above the reference group (less than high school), but especially for those with a college degree or higher. Those with a college degree or more have 6.49 times the assets compared with those who have less than a high school diploma. Being married is associated with greater wealth compared with those who are not married. Being Black or Hispanic is associated with decreased wealth, compared with Whites.

Perceived Stress. For the high perceived stress outcome (score of 3 or more), the models in Table 3 show that low childhood SES is a significant predictor of increased perceived stress in older adulthood. In the full model, those with low childhood SES have 32.4% increased odds of having high levels of perceived stress in late adulthood. Each level of educational attainment has a significant and negative association with perceived stress score. Having a college degree is protective of high perceived stress. Those with a college degree have 48.7% decreased odds of having a high perceived stress score in late adulthood. Wealth is also a significant predictor for decreased odds of high perceived stress.

Allostatic Load. Table 4 shows the incidence rate ratios for Poisson regression coefficients of allostatic load

(Waves 1 and 2) in adulthood on childhood SES. Results show that low childhood SES is associated with increased physiological dysregulation in older adulthood, but this relationship is explained primarily by educational attainment. Prior to controlling for education in Model 1, low childhood SES is a significant predictor of increased allostatic load in both Waves 1 and 2. In Model 2 for both waves, low childhood SES is no longer a significant predictor, suggesting education explains the association between low childhood SES and increased allostatic load. However, the relationship between education and allostatic load is stronger and more inclusive in Wave 1. In the full model for each wave (Model 3), having a college degree is significantly associated with decreased allostatic load, compared with the reference group. At Wave 1, those with a college degree have an allostatic load score 21.5% lower, on average, compared with individuals who have less than a high school diploma. At Wave 1, those with a high school diploma and those with some college also experience significantly lower allostatic load. Five years later in Wave 2, those with a college degree have an allostatic load score that is only 13.6% lower, on average, and being a high school graduate or having some college do not significantly differ from the reference group. In Wave 1, wealth is also a significant predictor of decreased physiological dysregulation, though to a lesser extent than a college education, and wealth is no longer a significant predictor in Wave 2. This suggests that the protective benefits of education, aside from a college degree, and wealth, may only last so long into late adulthood. Consistent with the literature, in all models, being Black is associated with significantly increased allostatic load score. Being married is significantly associated with lower allostatic load score, those to a lesser magnitude than education.

Longitudinal Allostatic Load Over 5 Years. Table 5 shows incidence rate ratios for the longitudinal allostatic load score over 5 years. In Model 1, those with low childhood

Table 3. Adult Outcomes, Logged Assets and High Perceived Stress^a.

	Logged Assets, OLS Regression Coefficients (SE)			High Perceived Stress, Odds Ratios (SE)		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Low childhood SES	-0.358*** (0.108)	-0.188 [†] (0.105)	-0.177 [†] (0.102)	1.346* (0.169)	1.324* (0.167)	1.324* (0.167)
Age	-0.011 (0.007)	0.003 (0.007)	0.013 [†] (0.007)	0.991 (0.008)	0.991 (0.008)	0.990 (0.009)
Female	-0.326** (0.107)	-0.249* (0.104)	0.018 (0.106)	1.303* (0.163)	1.272 [†] (0.160)	1.238 (0.163)
Race						
Black	-2.506*** (0.175)	-2.199*** (0.171)	-1.975*** (0.168)	1.043 (0.207)	0.831 (0.178)	0.822 (0.177)
Hispanic	-1.384*** (0.187)	-0.794*** (0.193)	-0.776*** (0.188)	1.260 (0.275)	1.165 (0.257)	1.168 (0.258)
Other	0.103 (0.335)	-0.039 (0.321)	-0.018 (0.313)	1.383 (0.518)	1.370 (0.516)	1.364 (0.515)
Adult SES						
High school graduate		0.755*** (0.180)	0.736*** (0.176)	0.755 (0.153)	0.810 (0.166)	0.808 (0.115)
Some college		1.178*** (0.172)	1.110*** (0.168)	0.688 [†] (0.133)	0.768 (0.152)	0.769 (0.152)
College graduate		1.878*** (0.182)	1.870*** (0.178)	0.433*** (0.094)	0.518** (0.116)	0.513** (0.115)
Assets					0.908** (0.028)	0.912** (0.029)
Married			0.966*** (0.114)			0.906 (0.130)

Abbreviations: OLS, ordinary least squares; SE, standard error; SES, socioeconomic status.

^aTable data are taken from the National Social Life, Health, and Aging Project, Waves 1 and 2. Standard errors are in parentheses. N = 1365.

[†]p ≤ .10. *p ≤ .05. **p ≤ .01. ***p ≤ .001.

Table 4. Poisson Regression Incidence Rate Ratios for Waves 1 and 2 Allostatic Load^a.

	Wave 1 (2005/2006)			Wave 2 (2010/2011)		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
Low childhood SES	1.079* (0.041)	1.053 (0.040)	1.048 (0.040)	1.076* (0.038)	1.060 (0.038)	1.056 (0.038)
Age	1.006* (0.003)	1.004 [†] (0.003)	1.003 (0.003)	0.997 (0.002)	0.996 [†] (0.002)	0.995* (0.002)
Female	1.094* (0.041)	1.084* (0.041)	1.050 (0.041)	1.032 (0.037)	1.028 (0.037)	0.992 (0.038)
Race/ethnicity						
Black	1.326*** (0.067)	1.265*** (0.068)	1.160*** (0.069)	1.314*** (0.062)	1.277*** (0.062)	1.202*** (0.063)
Hispanic	0.987 (0.067)	0.897 (0.064)	0.875 [†] (0.062)	1.015 (0.063)	0.960 (0.061)	0.947 (0.061)
Other	0.901 (0.109)	0.916 (0.106)	0.911 (0.102)	0.873 (0.118)	0.885 (0.117)	0.880 (0.113)
Adult SES						
High school graduate		0.849** (0.049)	0.869* (0.051)		0.928 (0.051)	0.941 (0.052)
Some college		0.857** (0.048)	0.891* (0.051)		0.893* (0.048)	0.915 (0.050)
College graduate		0.745*** (0.047)	0.785*** (0.052)		0.840** (0.048)	0.864* (0.052)
Assets(log)			0.972** (0.009)			0.985 [†] (0.009)
Perceived stress	1.012 (0.008)	1.007 (0.008)	1.004 (0.008)	1.007 (0.008)	1.028 (0.008)	1.001 (0.008)
Married			0.918* (0.038)			0.894** (0.036)

Abbreviation: SES, socioeconomic status.

^aTable data are taken from the National Social Life, Health, and Aging Project, Waves 1 and 2. Standard errors are in parentheses. N = 1365.

[†]P ≤ .10. *P ≤ .05. **P ≤ .01. ***P ≤ .001.

Table 5. Negative Binomial Regression Incidence Rate Ratios for Total Allostatic Load Change Score Over 5 Years^a.

	Model 1	Model 2	Model 3
Low childhood SES	1.076* (0.036)	1.056 (0.036)	1.051 (0.036)
Age	1.000 (0.002)	0.999 (0.002)	0.998 (0.002)
Female	1.043 (0.035)	1.034 (0.036)	0.998 (0.036)
Race/ethnicity			
Black	1.319*** (0.059)	1.279*** (0.059)	1.193*** (0.060)
Hispanic	1.006 (0.060)	0.936 (0.057)	0.917 (0.056)
Other	0.888 (0.107)	0.890 (0.103)	0.881 (0.098)
Adult SES			
High school graduate		0.897* (0.046)	0.912 [†] (0.047)
Some college		0.876** (0.043)	0.902* (0.045)
College graduate		0.801*** (0.042)	0.830*** (0.046)
Assets (log)			0.978* (0.009)
Perceived stress	1.009 (0.008)	1.005 (0.008)	1.002 (0.008)
Married			0.899** (0.033)

Abbreviation: SES, socioeconomic status.

^aTable data are taken from the National Social Life, Health, and Aging Project, Waves 1 and 2. Standard errors are in parentheses. N = 1365.

Total allostatic load score: scale created using indicators of risk for both waves, see Measures.

[†]P ≤ .10. *P ≤ .05. **P ≤ .01. ***P ≤ .001.

SES have 7.6% increased allostatic load over 5 years, all else being equal, compared with those who had average or above income in childhood. Similar to the singular time point allostatic load for Waves 1 and 2, once education is added to Model 2, low childhood SES is no longer a significant predictor. This suggests that education mediates this association. In all 3 models, Blacks have significantly higher longitudinal allostatic load over 5 years, compared with Whites. In the full model, Blacks

have 19.3% higher longitudinal allostatic load, on average, compared with Whites. Having some college education or a college degree are significantly associated with decreased longitudinal allostatic load over 5 years, compared with less than a high school diploma. The longitudinal allostatic load score is 17% lower, on average, for college graduates. Wealth is also a significant predictor of decreased longitudinal allostatic load score, but with a much smaller magnitude. Being married is

significantly associated with a 10.1% lower longitudinal allostatic load score.

Discussion

This study adds to existing literature on early life SES and its impact on physiological dysregulation in late adulthood. More specifically, it helps explain how the psychosocial stressors associated with low SES lead to the dysfunction of physiological systems over time. This, in turn, leads to increased risk of poor health outcomes and mortality. There are very few existing studies of older adults that include both information on early life SES and allostatic load as a measure of biological functioning. Using biological data eliminates some bias of alternative health measures, such as using respondent reported weight and blood pressure. Allostatic load also enables physiological dysregulation to be measured prior to reaching clinically significant levels and allows for a cumulative impact of SES on physiological dysregulation to be observed. The results suggest that childhood SES significantly affects adult outcomes, both directly and indirectly, even late into adulthood.

The first goal of our analyses was to examine the relationship between low childhood SES and adult outcomes: educational attainment, wealth, and perceived stress. We find that low childhood SES is significantly and directly associated with lower educational attainment, and this finding is consistent with the literature.^{34,44-47} Educational attainment, in turn, is significantly associated with household assets, perceived stress, and allostatic load in late adulthood. Low SES in childhood is only slightly associated with decreased household assets in late adulthood, with much of this relationship explained by educational attainment. Low childhood SES is significantly associated with increased risk of having high levels of perceived stress in late adulthood, and this relationship persists even after controlling for education and wealth.

The second goal of our analyses was to examine how childhood SES may directly and indirectly affect allostatic load at 2 separate time points. We found that this is an indirect relationship that is mediated mostly by education, with college education being especially important. In the first wave of measurement (2005/2006), all 3 education levels above the reference group were significantly associated with lower allostatic load, with a college education having the greatest effect. In the second measurement 5 years later, only a college education is protective against increased allostatic load, suggesting that all education levels may not experience the same length of health benefits in older ages. Wealth, in the form of assets, is a significant predictor of decreased

allostatic load in Wave 1, but is no longer significant in Wave 2. Again, this suggests that as individuals get older, adult SES has a somewhat smaller impact on late adult allostatic load. We were surprised to find that perceived stress was not a significant predictor of allostatic load for either wave, although low childhood SES was significantly associated with increased risk of high perceived stress. This may be due to the fact that allostatic load is a cumulative measure of physiological distress, whereas the perceived stress scale only asks about how the respondents felt in the past week.

Finally, the third goal of our analyses was to measure allostatic load longitudinally across 5 years. The benefit of the longitudinal allostatic load score measure is that it accounts for all indicators of biological risk, whether they increased, decreased, or stayed the same over 5 years. Those who go from showing high risk to no risk on an indicator from the first time point to the second are assigned a lower score than those who increase, which is consistent with the studies that show decreasing allostatic load score over time is associated with a decreased risk in mortality.^{24,25} And finally, it gives the highest score for individuals with high risk at both time points for an indicator, which theoretically suggests the greatest cumulative impact of wear and tear by maintaining a high-risk measure over 5 years. On the contrary, using the regular difference between scores over time may not properly shed light onto the cumulative allostatic load measure. Our findings for the total allostatic load change score scale are similar to the outcomes for Waves 1 and 2 allostatic load in that low childhood SES is a significant predictor of increased longitudinal allostatic load until education is added to the model. College education is especially important, but a college degree has a greater impact than some college. College graduates have 17% lower longitudinal allostatic load scores over 5 years, on average, compared with those without a high school diploma. Taken together, our findings underscore the importance of eliminating socioeconomic inequalities in educational attainment.

The policy implications from this study are focused on eliminating socioeconomic differences in educational attainment. There is evidence that public expenditures on education have a larger impact on state mortality rates than spending on housing, welfare, health, and hospitals.⁴⁸ In the practice sphere, the findings suggest that public and clinical health professionals should consider the effects of socioeconomic inequality and stress on physiological dysregulation in older individuals and provide access to key health-promoting and morbidity-preventing resources. Therefore, we recommend targeted practices in health services that give socioeconomically disadvantaged,

older adults access to resources that can help them reduce stress and monitor their health.

There are multiple strengths of this research, including the use of biomarkers, the longitudinal nature of the data, establishing a method of studying change in allostatic load, and including a measure of perceived stress. The NSHAP data set has biomarker indicators that are not available in many data sets, especially longitudinal ones. While there are multiple strengths to this research, it is not without its limitations. The primary limitations are the lack of childhood SES questions in Wave 1, only having 2 waves of data available, and missing biomarker measures. Analyses were run without individuals missing any biomarkers and showed similar results, but imputed data were chosen to boost statistical power. The lack of information on childhood SES prohibits us from directly observing its relationship with later morbidity and mortality. These are areas for future research once Wave 3 of the NSHAP becomes available. Future research should continue to include measures of early life exposures in order to better understand the complex relationship with late adult allostatic load. Most prior studies on early life SES and allostatic load in adulthood focus on middle adulthood, so they may not be observing the full picture if adult exposures only temporarily ameliorate negative early life exposures. Due to the complex nature of this relationship, future research should include advanced modeling techniques to fully illustrate how all these life course factors interact.

Author Contributions

KYG: Contributed to conception and design; contributed to acquisition, analysis, and interpretation; drafted manuscript; critically revised manuscript; gave final approval; agrees to be accountable for all aspects of work ensuring integrity and accuracy.

ACHN: Contributed to design; contributed to acquisition and interpretation; critically revised manuscript; gave final approval; agrees to be accountable for all aspects of work ensuring integrity and accuracy.

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References

- Galobardes B, Lynch JW, Davey Smith G. Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. *Epidemiol Rev.* 2004;26:7-21.
- Montez JK, Hayward MD. Early life conditions and later life mortality. In: Rogers RG, Crimmins EM, eds. *International Handbook of Adult Mortality*. Dordrecht, Netherlands: Springer; 2011:187-206.
- Zimmer Z, Hanson HA, Smith KR. Childhood socioeconomic status, adult socioeconomic status, and old-age health trajectories: connecting early, middle, and late life. *Demogr Res.* 2016;34:285-320.
- Shuey KM, Willson AE. Economic hardship in childhood and adult health trajectories: an alternative approach to investigating life-course processes. *Adv Life Course Res.* 2014;22:49-61.
- Willson AE, Shuey KM, Elder GH Jr. Cumulative advantage processes as mechanisms of inequality in life course health. *Am J Sociol.* 2007;112:1886-1924.
- Pearlin LI, Lieberman MA, Menaghan EG, Mullan JT. The stress process. *J Health Soc Behav.* 1981;22:337-356.
- Pearlin LI, Schieman S, Fazio EM, Meersman SC. Stress, health, and the life course: some conceptual perspectives. *J Health Soc Behav.* 2005;46:205-219.
- McLeod JD, Shanahan MJ. Trajectories of poverty and children's mental health. *J Health Soc Behav.* 1996;37:207-220.
- Read S, Grundy E. Allostatic load—a challenge to measure multisystem physiological dysregulation. http://eprints.ncrm.ac.uk/2879/1/NCRM_workingpaper_0412.pdf. Published September 2012. Accessed November 14, 2017.
- McEwen BS, Seeman T. Protective and damaging effects of mediators of stress: elaborating and testing the concepts of allostasis and allostatic load. *Ann N Y Acad Sci.* 1999;896:30-47.
- McEwen BS, Stellar E. Stress and the individual: mechanisms leading to disease. *Arch Intern Med.* 1993;153:2093-2101.
- Beckie TM. A systematic review of allostatic load, health, and health disparities. *Biol Res Nurs.* 2012;14:311-346.
- Seeman TE, Crimmins E, Huang MH, et al. Cumulative biological risk and socio-economic differences in mortality: MacArthur studies of successful aging. *Soc Sci Med.* 2004;58:1985-1997.
- Dowd JB, Palermo T, Chyu L, Adam E, McDade TW. Race/ethnic and socioeconomic differences in stress and immune function in the National Longitudinal Study of Adolescent Health. *Soc Sci Med.* 2014;115:49-55.
- Evans GW, Kim P. Childhood poverty and young adults' allostatic load: the mediating role of childhood cumulative risk exposure. *Psychol Sci.* 2012;23:979-983.
- Hawkey LC, Lavelle LA, Berntson GG, Cacioppo JT. Mediators of the relationship between socioeconomic status and allostatic load in the Chicago Health, Aging, and Social Relations Study (CHASRS). *Psychophysiology.* 2011;48:1134-1145.

17. Seeman TE, Merkin SS, Crimmins E, Koretz B, Charette S, Karlamangla A. Education, income and ethnic differences in cumulative biological risk profiles in a national sample of US adults: NHANES III (1988-1994). *Soc Sci Med*. 2008;66:72-87.
18. Howard JT, Sparks PJ. The role of education in explaining racial/ethnic allostatic load differentials in the United States. *Biodemography Soc Biol*. 2015;61:18-39.
19. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiol Behav*. 2012;106:29-39.
20. Merkin SS, Basurto-Dávila R, Karlamangla A, et al. Neighborhoods and cumulative biological risk profiles by race/ethnicity in a national sample of US adults: NHANES III. *Ann Epidemiol*. 2009;19:194-201.
21. Cohen AA, Milot E, Yong J, et al. A novel statistical approach shows evidence for multi-system physiological dysregulation during aging. *Mech Ageing Dev*. 2013;134:110-117.
22. Goldman N, Turra CM, Gleib DA, Lin YH, Weinstein M. Physiological dysregulation and changes in health in an older population. *Exp Gerontol*. 2006;41:862-870.
23. Juster RP, Marin MF, Sindi S, et al. Allostatic load associations to acute, 3-year and 6-year prospective depressive symptoms in healthy older adults. *Physiol Behav*. 2011;104:360-364.
24. Karlamangla AS, Singer BH, Seeman TE. Reduction in allostatic load in older adults is associated with lower all-cause mortality risk: MacArthur studies of successful aging. *Psychosom Med*. 2006;68:500-507.
25. Hwang AC, Peng LN, Wen YW, et al. Predicting all-cause and cause-specific mortality by static and dynamic measurements of allostatic load: a 10-year population-based cohort study in Taiwan. *J Am Med Dir Assoc*. 2014;15:490-496.
26. Milot E, Morissette-Thomas V, Li Q, Fried LP, Ferrucci L, Cohen AA. Trajectories of physiological dysregulation predicts mortality and health outcomes in a consistent manner across three populations. *Mech Ageing Dev*. 2014;141-142:56-63.
27. Upchurch DM, Stein J, Greendale GA, et al. A longitudinal investigation of race, socioeconomic status, and psychosocial mediators of allostatic load in midlife women: findings from the study of women's health across the nation. *Psychosom Med*. 2015;77:402-412.
28. Merkin SS, Karlamangla A, Diez Roux AV, Shrager S, Seeman TE. Life course socioeconomic status and longitudinal accumulation of allostatic load in adulthood: multi-ethnic study of atherosclerosis. *Am J Public Health*. 2014;104:e48-e55.
29. Dannefer D. Cumulative advantage/disadvantage and the life course: cross-fertilizing age and social science theory. *J Gerontol B Psychol Sci Soc Sci*. 2003;58:S327-S337.
30. DiPrete TA, Eirich GM. Cumulative advantage as a mechanism for inequality: a review of theoretical and empirical developments. *Annu Rev Sociol*. 2006;32:271-297.
31. O'Rand AM. Cumulative advantage theory in life course research. *Annu Rev Gerontol Geriatr*. 2002;22:14-30.
32. Ferraro KF, Shippee TP. Aging and cumulative inequality: how does inequality get under the skin? *Gerontologist*. 2009;49:333-343.
33. Ferraro KF, Shippee TP, Schafer MH. Cumulative inequality theory for research on aging and the life course. In: Bengtson VL, Gans D, Pulney NM, Silverstein M, eds. *Handbook of Theories of Aging*. New York, NY: Springer; 2009:413-433.
34. Ferraro KF, Kelley-Moore JA. Cumulative disadvantage and health: long-term consequences of obesity? *Am Sci Rev*. 2003;68:707-729.
35. Waite LJ, Laumann EO, Levinson W, et al. National Social Life, Health, and Aging Project (NSHAP). <http://www.icpsr.umich.edu/icpsrweb/NACDA/studies/20541/version/6>. Accessed November 14, 2017.
36. Kalimi M, Shafagoj Y, Loria R, Padgett D, Regelson W. Anti-glucocorticoid effects of dehydroepiandrosterone (DHEA). *Mol Cell Biochem*. 1994;131:99-104.
37. McEwen BS. Interacting mediators of allostasis and allostatic load: towards an understanding of resilience in aging. *Metabolism*. 2003;52(10 suppl 2):10-16.
38. Herd P, Karraker A, Friedman E. The social patterns of a biological risk factor for disease: race, gender, socioeconomic position, and C-reactive protein. *J Gerontol B Psychol Sci Soc Sci*. 2012;67:503-513.
39. Chonchol M, Katz R, Fried LF, et al. Glycosylated hemoglobin and the risk of death and cardiovascular mortality in the elderly. *Nutr Metab Cardiovasc Dis*. 2010;20:15-21.
40. Piazza JR, Almeida DM, Dmitrieva NO, Klein LC. Frontiers in the use of biomarkers of health in research on stress and aging. *J Gerontol B Psychol Sci Soc Sci*. 2010;65:513-525.
41. Nazare JA, Smith J, Borel AL, et al. Usefulness of measuring both body mass index and waist circumference for the estimation of visceral adiposity and related cardiometabolic risk profile (from the INSPIRE ME IAA study). *Am J Cardiol*. 2015;115:307-315.
42. Williams SR, McDade TW. The use of dried blood spot sampling in the national social life, health, and aging project. *J Gerontol B Psychol Sci Soc Sci*. 2009;64(suppl 1):i131-i136.
43. Cohen S, Kamarck T, Mermelstein R. A global measure of perceived stress. *J Health Soc Behav*. 1983;24:385-396.
44. Crystal S, Shea D. Cumulative advantage, cumulative disadvantage, and inequality among elderly people. *Gerontologist*. 1990;30:437-443.
45. O'Rand AM. The precious and the precocious: understanding cumulative disadvantage and cumulative advantage over the life course. *Gerontologist*. 1996;36:230-238.
46. Poulton R, Caspi A. Commentary: how does socioeconomic disadvantage during childhood damage health in adulthood? Testing psychosocial pathways. *Int J Epidemiol*. 2005;34:344-345.
47. Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet*. 2002;360:1640-1645.
48. Dunn JR, Burgess B, Ross NA. Income distribution, public services expenditures, and all cause mortality in US states. *J Epidemiol Community Health*. 2005;59:768-774.